Food and sex-specific growth strategies in a spider

Uhl, Gabriele ; Schmitt, Sandra ; Schäfer, Martin A ; Blanckenhorn, Wolf U

Abstract: Growth patterns are expected to differ between environments and between the sexes if there is sexual dimorphism, the general view being that male growth strategies are primarily sexually selected whereas female growth strategies are fecundity selected. We investigated the effects of food on sex-specific growth in the cellar spider Pholcus phalangioides, a sexually dimorphic spider with larger males than females. In a full sib design, 1164 offspring of 39 once-mated females were reared to sexual maturity under two feeding regimes. Food level had strong positive effects on (1) offspring body size, with males growing larger than females, and (2) offspring mass, with females maturing heavier than males; it had negative effects on (3) development time for males but not females. Males matured before females under unlimited food conditions. Analysing the entire ontogeny revealed that until the last instar, both sexes were equally retarded in development by food limitation, males lagging behind females. During the last instar, the picture reversed: development time of males was equally long at high and low food, while females had extremely long development at high food and abbreviated development at low food. We conclude that females are selected to increase mass and hence fecundity, while sexual selection apparently favours larger males but at the same time earlier maturity (i.e. protandry). Achieving both was only possible when food was plentiful, and is facilitated by a low genetic correlation between development time and body size. We found high genetic variation, as well as genotype–environment interactions, for size, mass, development time and growth rate, and consequently high full-sib but lower parent–offspring (size only) heritabilities. Genetic variation was not greater under food stress but genetic covariation was.

Originally published at:
Uhl, Gabriele; Schmitt, Sandra; Schäfer, Martin A; Blanckenhorn, Wolf U (2004). Food and sex-specific growth strategies in a spider. Evolutionary Ecology Research, 6:523-540.

Evolutionary Ecology Research, 2004, **6**: 523–540

Food and sex-specific growth strategies in a spider

Gabriele Uhl,[1]* Sandra Schmitt,[1] Martin A. Schäfer[1] and Wolf Blanckenhorn[2]

[1]*Department of Neuroethology, Institute of Zoology, University of Bonn, Endenicher Allee 11–13, 53115 Bonn, Germany and* [2]*Zoologisches Museum, Universität Zürich, Winterthurerstrasse 190, 8057 Zürich, Switzerland*

ABSTRACT

Growth patterns are expected to differ between environments and between the sexes if there is sexual dimorphism, the general view being that male growth strategies are primarily sexually selected whereas female growth strategies are fecundity selected. We investigated the effects of food on sex-specific growth in the cellar spider *Pholcus phalangioides*, a sexually dimorphic spider with larger males than females. In a full sib design, 1164 offspring of 39 once-mated females were reared to sexual maturity under two feeding regimes. Food level had strong positive effects on (1) offspring body size, with males growing larger than females, and (2) offspring mass, with females maturing heavier than males; it had negative effects on (3) development time for males but not females. Males matured before females under unlimited food conditions. Analysing the entire ontogeny revealed that until the last instar, both sexes were equally retarded in development by food limitation, males lagging behind females. During the last instar, the picture reversed: development time of males was equally long at high and low food, while females had extremely long development at high food and abbreviated development at low food. We conclude that females are selected to increase mass and hence fecundity, while sexual selection apparently favours larger males but at the same time earlier maturity (i.e. protandry). Achieving both was only possible when food was plentiful, and is facilitated by a low genetic correlation between development time and body size. We found high genetic variation, as well as genotype–environment interactions, for size, mass, development time and growth rate, and consequently high full-sib but lower parent–offspring (size only) heritabilities. Genetic variation was not greater under food stress but genetic covariation was.

Keywords: body size, development time, environmental stress, food limitation, genetic correlation, heritability, life history, sexual selection, sexual size dimorphism.

INTRODUCTION

In life-history theory, an individual's age and size at maturity is pivotal, as fitness is generally more sensitive to changes in these traits than to changes in any other trait (Roff, 1992; Stearns, 1992). Early maturation increases the probability of surviving to maturity

* Author to whom all correspondence should be addressed. e-mail: g.uhl@uni-bonn.de

and leads to a shorter generation time, whereas later maturation is beneficial if offspring number or quality increases with size and size is age dependent (Stearns, 1992). The benefits of early maturation are thus the costs of later maturation and vice versa, leading to a trade-off between body size and development time. Optimal size and age at maturity differ between species but also within species, as the size of males and females is influenced by different selection mechanisms. It is generally agreed that large female size is primarily fecundity selected and large male size primarily sexually selected (Blanckenhorn, 2000). In females, the number or quality of offspring increases strongly with body size (Clutton-Brock *et al.*, 1985; Shine, 1988; Honêk, 1993; Andersson, 1994). Thus, females should spend considerable time foraging and should delay maturation in favour of growth if resources are scarce. Males, on the other hand, primarily invest in obtaining mating partners and there is ample evidence for greater reproductive success of large males through male–male competition or female choice (Andersson, 1994). Differential selection on females and males is generally believed to cause differences in direction and degree of sexual size dimorphism (Elgar, 1992; Fairbairn, 1997).

Both theory and empirical evidence show that growth strategies are primarily shaped by: (1) food limitation, mediated by competition or predators, typically resulting in small body sizes (Stearns and Koella, 1986; Kozlowski, 1992); (2) time constraints on development due to seasonality if a specific life stage has to be reached in time (Roff, 1980; Rowe and Ludwig, 1991; Kozlowski, 1992); and (3) the mating system, as in species with first male sperm precedence early male maturation despite small size may increase the chances of mating with a virgin female. Thus, a relatively short time window for reproduction or a time-dependent change in reproductive value of the partners may counterbalance benefits of larger size and may result in protandry (Wiklund and Fagerström, 1977; Zonneveld, 1996). Fertilization advantage of the last male, on the other hand, very likely enhances size-dependent male–male competition for females (Andersson, 1994; Elgar, 1998).

Plastic responses in growth, body size and development time are generally predicted to be adaptive and evolve if genotypes are likely to encounter heterogeneous environments (Kozlowski, 1992; Roff, 1992; Stearns, 1992; Abrams *et al.*, 1996), which is by and large supported by the evidence (reviewed by Nylin and Gotthard, 1998). The effects of both food and time constraints on growth and final body size hinge on the trade-off between body size and development time (Roff, 1980). However, this does not have to be so, as there are two ways to get large: to grow for longer (i.e. increase development time) or to grow faster (i.e. increase growth rate). Early life-history models assumed growth rate to be generally maximized by (i.e. constant within) individuals, with growth being constrained primarily by external factors such as unfavourable temperatures or restricted food (e.g. Stearns and Koella, 1986). More refined later models incorporated variation in growth rate independent of development time (e.g. Abrams *et al.*, 1996). Growth is not always maximized because this either increases physiological mortality in response to environmental stress (e.g. Clutton-Brock *et al.*, 1985; Gotthard *et al.*, 1994; Blanckenhorn, 1998) or predation due to the riskier foraging necessary to achieve faster growth (e.g. Fraser and Gilliam, 1992; Werner and Anholt, 1993). Adaptive variation in growth rate implies and predicts that the phenotypic and, ultimately, the genetic correlation between body size and development time is weak, as in the simplest case growth rate is calculated as size accumulated per unit development time (Blanckenhorn, 1998). Analogously, the genetic correlation for these two traits between the sexes should be low in sexually dimorphic species, but this is not always the case (Preziosi and Roff, 1998).

Presumed trade-offs underlying the evolution of adaptive growth are likely to be detected only in resource-limited (i.e. stressful) environments, because when resources are not limited individuals can invest maximally in all traits. This means that laboratory studies attempting to demonstrate trade-offs have to test individuals in several environments. While this has been realized for some time (e.g. Hoffmann and Parsons, 1991), the corresponding experimental procedure has only recently become standard. Heterogeneous environments not only influence phenotypic but also genetic variation of quantitative traits, the common view being that genetic variation increases in unfavourable environments (Hoffmann and Parsons, 1991). However, reviewing the limited evidence on how genetic (co)variation changes with the environmental conditions, Hoffmann and Merilä (1999) concluded that the evidence to date is mixed and more work is needed to address the various conflicting hypotheses.

In spiders, growth is discontinuous with a fixed or slightly variable number of instars (Higgins and Rankin, 1996). The exoskeleton of the spider grows only during a moult (ecdysis), not between moults, and the magnitude of growth depends on the amount of reserves available (Homann, 1949). Thus spider growth is greatly affected by feeding rate, and in turn affects foraging through its influence on the potential prey spectrum (Vollrath, 1987). Female spiders are clearly fecundity selected, as larger size or mass results in a greater number of eggs laid (Peterson, 1950; Kessler, 1971; Fritz and Morris, 1985; Simpson, 1993), which may explain why female spiders are typically larger than males (Vollrath and Parker, 1992; Coddington *et al.*, 1997; Prenter *et al.*, 1998). Often male spiders undergo fewer moults until the adult stage than females, allowing them to mature earlier but at a smaller size (Schaefer, 1976; Elgar, 1992). The common sexual size dimorphism with smaller males observed in many spider species suggests that for males short maturation time is more important than size, whereas for females size is more important than short maturation time, as was shown for the lycosid *Hygrolycosa rubrofasciata* (Vertainen *et al.*, 2000).

Our study species *Pholcus phalangioides* (Pholcidae) is exceptional since males are slightly but significantly larger than females (Uhl, 1994a). In this species, large males have a competitive advantage over small males (Schaefer and Uhl, 2003), and females are fecundity selected as large body size results in a higher number of eggs per clutch (Uhl, 1998). Last male sperm precedence is strong when females are mated with two males in succession. However, only 68% of females re-mated in a double mating trial, resulting in a considerable advantage for first males (Schäfer and Uhl, 2002). We would therefore predict that male size is under strong selection, and that males of this species consequently have longer development times than females or faster growth rates to reach a larger size sooner, should protandry be beneficial.

In this study, we examined the growth of male and female spiders under conditions of limited and unlimited food, and analysed the effects of treatment on development time, body size and mass at adulthood of spiders reared from the egg sac. For a plastic trait to respond to selection and evolve, there has to be genetic variance in the traits as well as in the reaction norm. We therefore analysed among-family variation using a full-sib quantitative genetic design, generating estimates of heritabilities and genetic correlations for (and between) the sexes in both food environments. A comprehensive quantitative genetic study of spider growth and development is lacking, largely because of prohibitively long development times and practical problems with rearing most species in the laboratory. We specifically address the prediction that *P. phalangioides* adaptively vary their growth rate and that consequently the phenotypic and genetic correlation between size and develop-

ment time should be weak. We also assess whether genetic variation and covariation is indeed greater in the stressful food environment (cf. Hoffmann and Parsons, 1991; Hoffmann and Merilä, 1999).

MATERIALS AND METHODS

Spider holding conditions

Juvenile spiders were collected from populations in and around Bonn, Germany, in spring 2001. Spiders were kept individually in plastic containers ($10 \times 10 \times 5$ cm) at room temperature and natural photoperiod. Once a week they were fed with about 50 *Drosophila*, and water was supplied. During the final inter-moult interval, the spiders were fed 10–15 *Drosophila* per week plus either one maggot of *Lucilia* spp. or one *Gryllus domesticus* of about 1 cm. All spiders thus received the same amount and type of food to minimize effects of maternal environment. After the animals reached sexual maturity, the length of tibia plus patella of the first pair of legs was measured with an ocular micrometer on anaesthetized animals to the nearest 0.1 mm. Leg length is a good measurement of overall body size in both sexes, as it was shown to be isometrically correlated with prosoma length and width; however, the repeatability of leg measurements is higher (Uhl, 1994a). The legs of females used to form mating pairs were 11.00 ± 0.90 (mean ± standard deviation) mm long (range 9.14–13.65 mm, $n = 40$) and those of males 10.99 ± 0.89 mm (range 9.04–13.28, $n = 40$). We avoided matings between spiders of very different sizes to minimize possible effects of genital mismatch (Uhl, 1994b). Matings were staged as described in Schäfer and Uhl (2002). Thirty-nine females copulated once with an inexperienced male and were subsequently kept separately until oviposition. There were thus 39 full-sib families.

Rearing procedures of offspring

On average, 30.31 ± 2.15 (mean ± standard error) spiderlings hatched from the first clutch of 39 females, 1182 of which were reared to adulthood. The time period from hatching to the final moult was taken as total development time. Immediately after hatching spiderlings do not feed. After the first moult, spiderlings were removed from their mother's box and each individual was transferred to a separate container (10 cm high, 5 cm in diameter). They were then kept in a climate chamber at 23.5°C and with a 14 light : 10 dark photoperiod according to Miyashita (1988). Average humidity was $60 \pm 2\%$.

Each full-sib family of spiderlings was divided randomly among two feeding levels. High food level spiderlings received about three times more food than low food level spiderlings, comparable to the feeding regime applied by Vertainen *et al.* (2000). High food level spiderlings received four *Drosophila* and some drops of water three times per week until the third instar. After the third moult, spiderlings received five *Drosophila* three times per week plus one *Tribolium castaneum* larva once a week. Low food spiderlings received three *Drosophila* and water once a week only, and after the third moult five *Drosophila* per week. *Drosophila* were raised on a nutrient-rich medium following Mayntz and Toft (2001), who found that this medium resulted in a survival rate of 60% in a lycosid, which is unusually high for spiders. *Tribolium castaneum* larvae were added in the high food treatment to offer a more diverse diet, as a mixed diet has been shown to affect survival of spiderlings in previous studies on pholcid and lycosid spiders (Jakob and Dingle, 1990; Uetz *et al.*, 1992).

We checked spiders daily for moults and death. Altogether, 1164 of the 1182 offspring reached adulthood. Thus mortality was only 1.5%. After the final moult to adulthood, we determined sex, size and mass; 583 females and 581 males were determined. For the low feeding regime, we had one family from which no females matured and one family from which no males matured. Adult offspring size was measured to the nearest 0.1 mm with an ocular micrometer, and wet body mass was determined on a Mettler H11w scale with 0.01 mg accuracy. Although body size and mass were found to be significantly correlated (Schäfer and Uhl, 2002), optimal strategies in these two traits may differ between males and females, which cannot be tested using only one parameter as in previous studies (e.g. Jakob and Dingle, 1990; Simpson, 1995; Vertainen *et al.*, 2000).

Statistical analyses were performed with SPSS 10.0. Two-tailed tests were applied throughout and alpha was set at 0.05. We used univariate analyses of variance (ANOVA) to examine effects of food level, sex (fixed factors) and family (random factor) on life-history parameters (size, mass, development time), similar to Fry (1992). We did not use a nested design, as the number of male and female offspring per treatment within each family varied. In spiders, sex cannot be assessed until the last instar; thus small spiderlings were divided into two treatment groups without knowledge of their future sex. However, when using such a design on a balanced subset of the data, qualitatively similar results were obtained. Data on development time were not normally distributed, which could not fully be corrected by log transformation, but ANOVA results tend to be robust to such small deviations at sample sizes as large as obtained here (Sokal and Rohlf, 1995).

We calculated heritabilities based on full-sibs as $h^2 = 2V_{family}/(V_{family} + V_{error})$ following Roff (1997), where V_{family} is the among-family variance component and V_{error} is the within-family (error) variance component, separately for all treatment combinations (sex and food) because variances differed. Approximate standard errors (SE) were supplemented as given in Becker (1992). Significant genetic variation is indicated by significant family effects in the ANOVA and $2SE(h^2)$ not overlapping zero. Family sizes were unequal, so the weighted mean family size k is reported (Becker, 1992; Roff, 1997). Variance components were calculated using restricted maximum likelihood in SPSS 10 (procedure varcomp; however, other methods yielded very similar estimates). We also performed parent–offspring regressions for leg length, separately for the sexes and treatments, yielding an estimate of the narrow-sense heritability (possibly including parental effects) for body size (Roff, 1997).

To assess the trade-off between development time and body size or mass (within individuals), we calculated genetic correlations in two ways, separately for all treatment combinations. First, pairwise genetic correlations were calculated from analyses of covariance (ANCOVA) analogous to the ANOVAs for the heritability calculations, deriving the covariance from the mixed-model ANOVA and the variances from separate ANOVAs (Falconer, 1989; Becker, 1992; Roff, 1997). An approximate standard error for this correlation, r_g, is given in Roff (1997, p. 81) as

$$SE(r_g) = (1 - r_g^2)\sqrt{\frac{SE(h_X^2)SE(h_Y^2)}{2h_X^2h_Y^2}}$$

where h_X^2 and h_Y^2 refer to the heritabilities of the two variables X and Y. Second, the genetic correlation can also be approximated as the Pearson product–moment correlation between

the family trait means, r_m (Via, 1984; Roff, 1995, 1997), with its standard error being that of any phenotypic correlation, r_p, among n pairs of variables:

$$SE(r_p) = \sqrt{\frac{1 - r_p^2}{n - 2}}$$

Using these standard errors, significant deviations of both r_g and r_m from zero and unity can be found using one-sample t-tests. The corresponding phenotypic correlations, $r_p = COV(X,Y)/(SD(X)SD(Y))$, where COV and SD refer to covariance and standard deviation respectively, were also calculated.

Family-mean genetic correlations between the sexes were analogously calculated using the family mean (yielding $r_{m(sex)}$) and the ANCOVA (yielding $r_{g(sex)}$) methods, separately for both food environments. The ANCOVA method considers the trait as expressed in two environments, the 'environment' here being sex (Fry, 1992; Roff, 1997, p. 89). No standard error formula is available. However, the F-ratio of the among-family and the family × sex interaction mean-squares in this mixed model serves as an approximate test of whether $r_{g(sex)}$ is significantly greater than zero (Fry, 1992; Roff, 1997, p. 90).

RESULTS

Offspring size

The mean (± standard deviation) size of well fed females was 9.97 ± 0.72 mm (n = 297) and of poorly fed females 9.59 ± 0.65 mm (n = 286). Similarly, male offspring in the high food condition were larger (10.34 ± 0.76 mm; n = 285) than those in the low food condition (9.88 ± 0.73 mm; n = 296; Fig. 1). Offspring size thus increased with feeding level, equally for males and females (sex × food level interaction; not significant, NS) and males grew significantly larger than females (Table 1). Furthermore, there was a strong family effect on offspring size, and a significant interaction between family and feeding regime indicating genotype–environment interactions across food environments (Fry, 1992; Table 1).

Offspring mass and condition

Feeding treatment also had a significant effect on offspring mass (Table 1). Well fed females weighed 21.43 ± 3.94 mg and poorly fed females 20.42 ± 3.45 mg, while well-fed males weighed 19.98 ± 3.64 mg compared with 18.50 ± 3.09 mg for males in the low food condition (Fig. 1). Female offspring were significantly heavier than male offspring under both feeding regimes, reversing the size dimorphism obtained for leg length. The response to food limitation was slightly stronger for males than for females, as indicated by the marginally significant sex × food level interaction (Table 1, Fig. 1). There was a strong family effect and a significant interaction between family and food regime (Table 1). The significant sex × family interaction indicates varying degrees of size dimorphism across families.

We also calculated offspring condition as size divided by mass for the benefit of some readers, and found that male condition was significantly lower than female condition under both feeding regimes (females, high food: 2.14 ± 0.30; females, low food: 2.13 ± 0.31; males, high food: 1.92 ± 0.23; males, low food: 1.86 ± 0.22). The corresponding analysis of

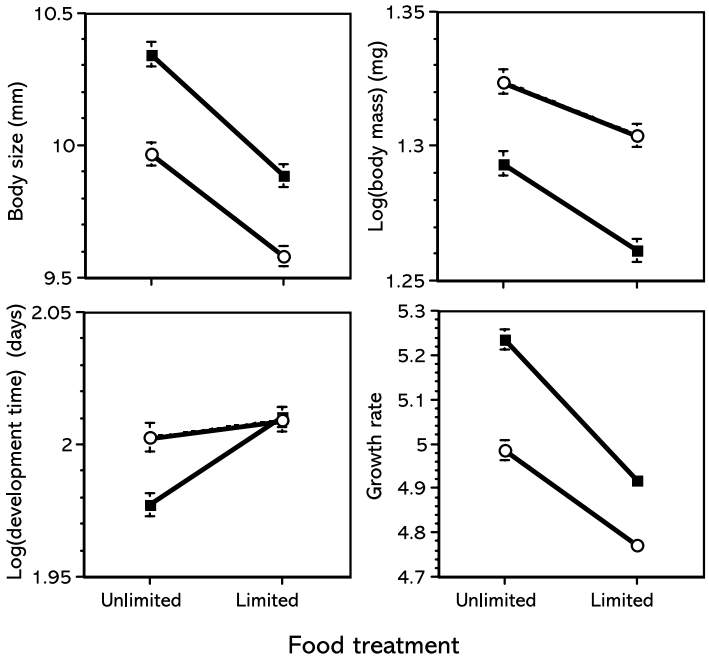

Fig. 1. Population reaction norms (± SE) of adult offspring traits for female (○) and male (■) *Pholcus phalangioides* in the limited and unlimited food conditions (body size = length of patella and tibia of the right first leg in mm; growth rate = body size at adulthood/log(development time)).

variance of mass with size as covariate (Jakob *et al.*, 1996) revealed: (1) a marginally significant effect of food; (2) a significant effect of sex, with females exhibiting higher condition than males; and (3) a significant sex × food interaction, with females responding less to food limitation than males (Table 1). We also found significant genetic effects and significant phenotype–environment interactions (Table 1).

Offspring development time and growth rate

All 1164 offspring took five moults from hatching to adulthood. Interestingly, food shortage significantly prolonged development time only in males. Males in the high food condition matured after 96.43 ± 20.12 days, while males in the low food condition matured after 103.70 ± 19.82 days. In contrast, well-fed females matured as fast (103.48 ± 27.35 days) as poorly fed females (103.96 ± 22.51 days; Fig.1), resulting in a food × sex interaction and an overall non-significant effect of sex on development time (Table 1). There were again significant family effects and a significant interaction between family and feeding regime indicating heritable phenotypic plasticity (Table 1).

Although somewhat redundant, and imperfect given typically non-linear growth trajectories (e.g. Blanckenhorn, 1998; Badyaev *et al.*, 2001), we additionally analysed growth rate as size/log(development time). Males had faster growth rates than females under both feeding regimes with an expected negative effect of low food on growth rate (well-fed females 4.99 ± 0.40, poorly fed females 4.77 ± 0.31, well-fed males 5.24 ± 0.40, poorly fed males 4.92 ± 0.32) (Fig. 1). Growth rates differed between families, and families

Table 1. Results of full-sib ANOVA for the effects of feeding regime (food), sex and family on offspring body size (leg measure), body mass (log), condition (ANOVA as before with size as covariate), development time (days from hatching until final moult, log), and growth rate (size/log(development time)) ($n = 1164$)

		Body size		Body mass (log)		Condition		Development time (log)		Growth rate	
Variable	d.f.	MS	*F*	MS	*F*	MS	*F*	MS	*F*	MS	*F*
Food[a]	1	37.96	83.90***	0.142	19.25***	0.016	3.64(*)	0.079	6.78***	14.96	86.54***
Sex[b]	1	27.91	98.72***	0.297	46.81***	0.826	284.95***	0.012	2.20	8.37	86.73***
Food × sex[c]	1	0.03	0.16	0.014	3.61(*)	0.012	4.72*	0.029	5.75***	0.251	3.88(*)
Family[d]	38	8.80	13.84***	0.064	5.47***	0.012	2.30*	0.055	3.50***	1.997	8.13***
Family × food[e]	38	0.52	2.74**	0.008	2.19**	0.005	1.82*	0.014	2.65**	0.203	3.09***
Family × sex[f]	38	0.30	1.60(*)	0.007	1.85*	0.003	1.28	0.006	1.12	0.107	1.61(*)
Family × sex × food[g]	38	0.19	0.90	0.004	1.14	0.002	1.25	0.005	1.21	0.066	1.08
Error	1010	0.21		0.003		0.002		0.004		0.061	

***$P < 0.001$; **$P < 0.01$; *$P < 0.05$; (*) $P < 0.1$.

Corrected error terms:
[a] 0.787 MS(food × family) + 0.213 MS(error).
[b] 0.792 MS(sex × family) − 0.005 MS(sex × food × family) + 0.213 MS(error).
[c] 0.787 MS(sex × food × family) + 0.213 MS(error).
[d] MS(sex × family) + 1.017 MS(food × family) − 1.017 MS(sex × food × family).
[e] MS(sex × food × family).
[f] 1.017 MS(sex × food × family) − 0.017 MS(error).
[g] MS(error).

showed a plastic response to different food levels as indicated by the significant food × family interaction (Table 1). The interactions food × sex and family × sex were only marginally significant. If growth rate was calculated as mass per log(development time), the results only differed from the previous ones in that the interaction between food and sex was significant ($P = 0.031$), with females responding less strongly to food level than males.

Duration of instars

Figure 2 demonstrates that the durations of the five instars vary considerably, the last instar leading to adulthood being almost twice as long as the preceding instar and with triple the variance. To determine at which instar differences between the sexes and food levels become apparent, we performed a multivariate analysis of variance. Durations of instars one to five were taken as dependent variables, food level and sex as fixed factors, and family as a fixed blocking variable to remove genetic effects (Table 2). To avoid redundancies, we report average results based on family means.

There was a significant food level effect on instars two and three and a marginal effect on instar four. In all cases, development time was longer at low food; in instar five this was reversed, but not significantly so (Table 2; Fig. 2). Males had longer development times during the first four instars (significant only in the third and marginally in the first). This was reversed in the fifth instar, largely because females in the high food condition showed extremely prolonged development compared with the other three treatment combinations (which were equal), resulting in a significant interaction between food level and sex (Fig. 2).

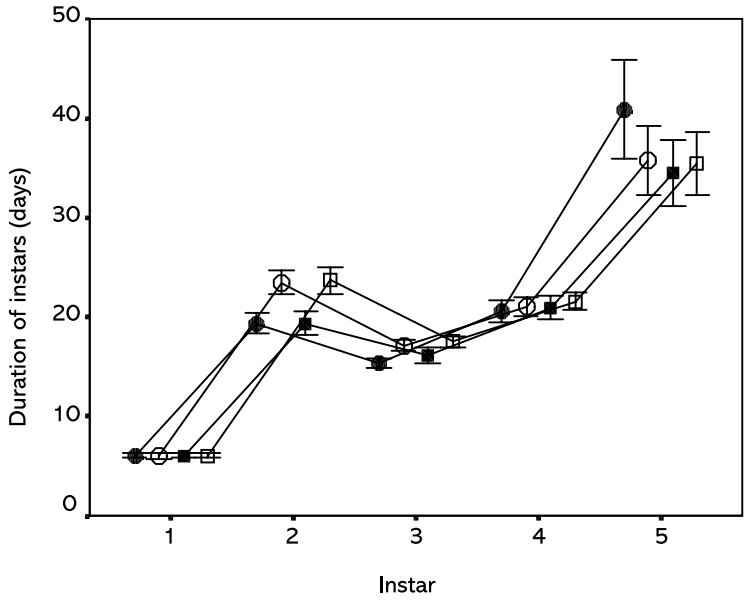

Fig. 2. Duration of instars 1–5 (from hatching until the final moult to adulthood, ± SE) for females (circles) and males (squares) in the low (white) and high (black) food conditions.

Table 2. Effect of food and sex on mean duration of instars

		Food		Sex		Food × Sex		Error	
Variable	d.f.	MS	*F*	MS	*F*	MS	*F*	d.f.	MS
Instar 1	1	0.00	0.00	0.03	2.99(*)	0.00	0.00	112	0.01
Instar 2	1	689.6	135.4***	0.01	0.00	0.19	0.036	112	5.09
Instar 3	1	87.24	36.09***	15.10	6.24*	2.35	0.974	112	2.42
Instar 4	1	17.13	3.81(*)	6.92	1.54	0.31	0.069	112	4.49
Instar 5	1	156.0	2.19	440.8	6.18*	381.1	5.345*	112	71.29

Note: Univariate output of a multivariate analysis of variance with instars 1–5 as independent variables, food and sex as fixed factors and family as covariate.

Heritabilities

Estimated heritabilities based on full sibs were highest for body size and somewhat lower for body mass, development time and growth rate (not shown), and were of similar magnitude in the sexes and at both food levels (Table 3). The latter resulted because (except perhaps for female size) genetic and environmental (= error) variance decreased equally at low food (Table 3).

For body size only, we could also calculate (narrow-sense) heritabilities based on a parent–offspring regression. As expected because of dominance, maternal and common environment effects (Roff, 1997), these estimates were considerably lower than those based on full sibs, whereby son–father estimates tended to be higher (son–father at high food: $h^2 = 0.44 \pm 0.25$ (SE), $P = 0.081$; son–father at limited food: $h^2 = 0.48 \pm 0.23$, $P = 0.040$; daughter–mother at high food: $h^2 = 0.14 \pm 0.20$, $P = 0.499$; daughter–mother at limited food: $h^2 = 0.23 \pm 0.20$, $P = 0.267$).

Phenotypic and genetic correlations

Phenotypic and family-mean correlations between body size measured as leg length and adult body mass (log-transformed) were similarly high and significant in all treatment groups (females at high food: $r_p = 0.77$ and $r_m = 0.79$; females at low food: $r_p = 0.52$ and $r_m = 0.62$; males at high food: $r_p = 0.87$ and $r_m = 0.89$; males at low food: $r_p = 0.76$ and $r_m = 0.62$; all $P < 0.001$, $n = 285$ or 296 individuals and $N = 38$ or 39 families).

We found only slight, albeit significant positive correlations between body size and development time (Fig. 3). The phenotypic correlations were $r_p = 0.12 \pm 0.06$ ($P < 0.05$; females at high food), 0.36 ± 0.06 ($P < 0.001$; females at low food), 0.15 ± 0.06 ($P < 0.05$; males at high food) and 0.44 ± 0.05 ($P < 0.001$; males at low food; $n = 285$ to 296 individuals). The correlations were higher at low than at high food (significant food × log(development time) interaction in an ANCOVA with body size as the dependent variable, sex and food as fixed factors and log development time as covariate: $F_{1,1156} = 25.97$, $P < 0.001$), and also higher for males than females (sex × log(development time) interaction: $F_{1,1156} = 5.59$, $P = 0.018$). This occurred because the phenotypic variation in both traits (in the denominator) was reduced while the covariance between both traits (in the numerator; see formula given above) increased at low food. The corresponding full-sib

Table 3. Full-sib $h^2 \pm$ SE, the corresponding variance components (V_{family}: among-family variance component and V_{error}: within-family variance component), and the family-mean genetic correlation between the sexes calculated from family means, $r_{m(sex)} \pm$ SE, or with the ANCOVA method, $r_{g(sex)}$, for three traits at high and low food in the spider *Pholcus phalangioides*

	Full-sib h^2			V_{family}; V_{error}	
	Males	Females	$r_{m(sex)}$; $r_{g(sex)}$	Males	Females
Body size					
High food	1.22 ± 0.13*** $N = 39$; $k = 7.26$	1.00 ± 0.14*** $N = 39$; $k = 7.57$	0.89 ± 0.08; 0.98***	0.345; 0.221	0.240; 0.238
Low food	1.23 ± 0.13*** $N = 38$; $k = 7.54$	1.15 ± 0.13*** $N = 38$; $k = 7.29$	0.84 ± 0.09; 0.96***	0.323; 0.202	0.243; 0.181
Development time				($\times 10^{-3}$)	($\times 10^{-3}$)
High food	0.64 ± 0.14*** $N = 39$; $k = 7.26$	0.67 ± 0.14*** $N = 39$; $k = 7.57$	$0.71 \pm 0.12^{\#}$; 0.95***	1.71; 3.63	3.21; 6.36
Low food	0.60 ± 0.13*** $N = 38$; $k = 7.54$	0.51 ± 0.14*** $N = 38$; $k = 7.29$	$0.64 \pm 0.13^{\#}$; 0.81***	1.23; 2.88	1.38; 4.05
Body mass				($\times 10^{-3}$)	($\times 10^{-3}$)
High food	0.98 ± 0.14*** $N = 39$; $k = 7.26$	0.70 ± 0.14*** $N = 39$; $k = 7.57$	$0.78 \pm 0.11^{\#}$; 0.94***	3.04; 3.19	2.01; 3.73
Low food	0.88 ± 0.14*** $N = 38$; $k = 7.54$	0.68 ± 0.14*** $N = 38$; $k = 7.29$	$0.53 \pm 0.15^{\#}$; 0.84***	2.36; 2.97	1.56; 3.54

Note: Genetic estimates are based on logarithm for mass and development time.
N = number of families; k = corrected family size according to Roff (1997). ***$P < 0.001$; $^{\#}r_m$ different from one at $P < 0.05$.

family mean correlation estimates, r_m, between development time and size as well as mass were very similar, although not significant due to the lower sample size except for males reared in the low food condition ($n = 38$–39 families; Fig. 3): $r_p = 0.11 \pm 0.16$ (NS; females at high food), 0.22 ± 0.16 (NS; females at low food), 0.06 ± 0.16 (NS; males at high food) and 0.42 ± 0.15 ($P < 0.01$; males at low food). Again, the food × log(development time) interaction in an ANCOVA was significant ($F_{1,35} = 4.39$, $P = 0.044$), indicating higher genetic correlations in the limited food condition, but the sex × log(development time) interaction was not ($P > 0.1$). However, the corresponding genetic correlations, r_g, calculated from ANCOVAs were higher and significant at low food but nil for high food: $r_g = 0.03 \pm 0.11$ (NS; females at high food), 0.49 ± 0.09 ($P < 0.001$; females at low food), 0.16 ± 0.10 (NS; males at high food) and 0.70 ± 0.05 ($P < 0.001$; males at low food; n as above). Although this approximate SE(r_g) (see Methods) tends to be an underestimate (Roff and Preziosi, 1994), the r_g at low food are so high that they are unlikely to become non-significant. The corresponding situation for body mass and development time was qualitatively similar, only that the corresponding correlations (both log-transformed) were even lower and generally nil except for males at low food ($r_p = 0.28$, $P < 0.001$).

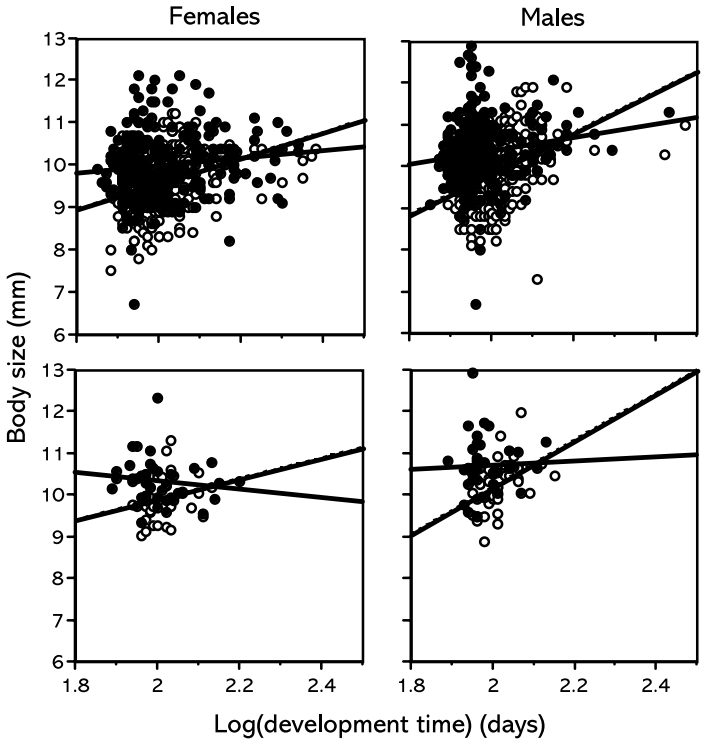

Fig. 3. Phenotypic (r_p, top) and family-mean genetic (r_m, bottom) correlations between body size and log(development time) for females (left) and males (right) in the limited (white circles and broken line) and unlimited (black circles and unbroken line) food conditions.

The genetic correlations between the sexes based on the family mean method, $r_{m(sex)}$, were moderate to high and significantly less than one for mass and development time, but not size (Table 3). The corresponding correlations calculated from ANCOVAs, $r_{g(sex)}$, were generally even closer to one (Table 3). Non-significant family × sex interactions (as for condition and development time in Table 1) suggest that $r_{g(sex)}$ is perhaps not different from one, although this was not specifically tested. All these correlations between the sexes are significantly greater than zero, however, based on the appropriate mixed model *F*-test for $r_{g(sex)}$ (see Methods; Table 1) or the family mean SE for $r_{m(sex)}$.

DISCUSSION

Male and female *Pholcus phalangioides* do not differ in the number of instars. Differences in size must therefore result from differential changes at ecdysis, reflecting weight gain of each instar (Schaefer, 1976; Higgins and Rankin, 1996; Hutchinson *et al.*, 1997). The strong effect of food level on size and mass of *P. phalangioides* suggests that the largest part of variance in adult size results from feeding activity during the juvenile phase. Interestingly, the direction of sexual size dimorphism depends on the trait considered: *P. phalangioides* males grow larger (in terms of leg length) but females gain more body mass, under both high

and low food conditions. As a consequence, size and mass should not be used synonymously in life-history studies, despite the fact that they are highly correlated.

Our results show that reaction norms can differ between males and females in unanticipated and complex ways. Males reared in the low food condition had decreased size and mass and prolonged development time, a response in line with most theory (e.g. Stearns and Koella, 1986). Females in the low food condition also showed a strong reduction in size and a less pronounced reduction in mass. Interestingly, however, females showed little response in overall development time (Fig. 1). That is, male *P. phalangioides* appeared unable to increase growth rates under food stress to the same extent as females, thus paying a cost. Furthermore, females achieve similar body condition under both feeding regimes but male condition decreases under food stress. Comparative data suggest that males paying higher costs than females is a general phenomenon in insects (Signorelli, 2002), but the phenomenon and its causes have to be explored further. Our data on duration of instars add even more complexity to the picture. In essence, until the last instar both sexes were equally retarded in development by food limitation (i.e. they had longer development times), with males lagging behind females. During the longest, ultimate juvenile instar, however, this picture reversed: males abbreviated their development equally in the high and low food conditions, while females had extremely long development at high food and abbreviated development at low food, producing the interaction between food and sex for total development time shown in Fig. 1. Abbreviated male development relative to females suggests that protandry is advantageous in this species (discussed further below). At the same time, prolonged female development indicates that females invest primarily in weight and size gain to achieve higher fecundity when food is abundant (but not when food is limited). The reversed sexual dimorphism for body size (leg length) and mass also suggests that female growth is aimed at increasing mass while male growth is aimed at increasing size. However, it is difficult to understand why fifth-instar females shortened their development in the low food condition, whereas before it was prolonged relative to the high food treatment. Perhaps the time window for successful reproduction is more restricted for females than assumed, leading to a compensation in development time during the last instar. Reversals in growth response during ontogeny as found here occur in other species as well (e.g. birds: Badyaev *et al.*, 2001; insects: Klingenberg, 1998; Fischer and Fiedler, 2000, 2001) and demonstrate a high degree of developmental plasticity relating to the ontogeny of sexual size dimorphism. Differential growth responses of the sexes are not built into most theoretical models (e.g. Stearns and Koella, 1986) but they should be, as they may be a general phenomenon calling for more detailed investigation.

In the high food condition, males not only matured at a larger size but also 7 days earlier than females. This suggests that although size plays an important role in male–male competition (Schaefer and Uhl, 2003), maturing early is a further advantage for males of this species. Finding a virgin female and defending her for only a few hours after copulation can confer exclusive fertilization success to a male, as once mated females do not necessarily re-mate (Schäfer and Uhl, 2002) and re-mating probability decreases rapidly with the inter-mating interval (G. Uhl, A. Dietzsch and M. Schäfer, unpublished data). Thus, while male–male competition selects for large male size (Schaefer and Uhl, 2003), low female re-mating probability selects for faster male development, both of which apparently can only be realized when food is abundant. This shows that the sexual size dimorphism of a species is the end result of a complex interplay of sex-specific advantages and disadvantages of large body size (Fairbairn, 1997; Blanckenhorn, 2000). In spiders, we clearly need

more life-history and sexual selection data, particularly for species exhibiting the 'normal' female-biased sexual size dimorphism.

Our results demonstrate significant genetic (i.e. among-family) variation for body size, body mass, development time and growth rate. This agrees with a study on the related spider *Holocnemus pluchei*, for which a family effect on development time was demonstrated in an ANOVA (without calculating heritabtilies: Jakob and Dingle, 1990). Our full-sib heritabilities were quite high (>0.6) and thus probably contain significant non-additive (dominance), maternal and common-environment effects, as the parent–offspring heritability (for body size only) was considerably lower, albeit significant and in line with estimates from other species (Mousseau and Roff, 1987). A full-sib design does not control for non-additive effects. Early maternal effects via egg size or quality are probably present but limited, since female body size translates into increased egg number rather than egg weight in a related spider (Skow and Jakob, 2003). Also, spiderlings were not fed by their mother and they did not feed themselves during the first instar while they were kept together. However, carry-over environmental or genotype × environment effects cannot be completely ruled out here, as parents were collected from the wild as juveniles but kept under the same conditions thereafter. Furthermore, common environment effects were minimized by rearing spiderlings in individual containers.

We also found significant interactions between family and feeding regime on size, mass, growth and development that reveal heritable phenotypic plasticity, indicating that some families did better in the poor food condition than others (cf. Via, 1984). High genetic variation despite persistent directional selection is known for condition-dependent expression of sexually selected (Grafen, 1990; Pomiankowski and Møller, 1995; Rowe and Houle, 1996; David *et al.*, 2000; Blanckenhorn and Hosken, 2003) as well as life-history traits (Houle, 1992; Blanckenhorn, 1998).

The positive correlation between development time and body size, which features as a key assumption in many life-history models (Roff, 1980, 1992; Rowe and Ludwig, 1991), is weak to non-existent in *P. phalangioides*. This holds for both phenotypic and genetic correlations, which were quite similar (as is the case in general: cf. Roff, 1995, 1996). This is perhaps not surprising, as extensive adaptive phenotypic plasticity in growth, as exemplified here, requires this correlation to be low – that is, functionally unconstrained (Abrams *et al.*, 1996; Blanckenhorn, 1998; cf. Roff, 2000). The correlations were similar for the sexes, but were greater when resources were limited. This supports the most common view that stress conditions increase phenotypic differences between individuals and hence genetic (co)variation (Hoffmann and Parsons, 1991; Hoffmann and Merilä, 1999). However, heritabilities were similar for the sexes and both food levels, largely because environmental and genetic variance decreased in conjunction (cf. Blanckenhorn, 2002). The issue of whether and how genetic estimates vary across environments thus remains complex, as it seems to depend on the environment, trait and species under consideration. This is perhaps not surprising given the large number of genetic mechanisms suggested to affect genetic (co)variance (Mousseau and Roff, 1987; Roff, 1995, 1996; Hoffmann and Merilä, 1999). Our results show that even moderate food limitation that does not affect mortality can lead to significant changes in phenotypic and genetic variation.

Although *P. phalangioides* is sexually dimorphic and growth strategies differ between the sexes, we found strong genetic correlations between the sexes for all traits assessed. For some traits (body size based on $r_{m(sex)}$ and condition and development time based on $r_{g(sex)}$) they were not significantly lower than one, leading to the conclusion that genetic constraints

preventing separate evolution of the sexes are currently considerable. Somewhat paradoxically, such functional constraints can be strong even in sexually dimorphic species (e.g. Preziosi and Roff, 1998), although this does not preclude such genetic correlations having been lower at some earlier stage of size dimorphism evolution.

In conclusion, we found highly flexible and complex growth strategies of male and female cellar spiders in response to food shortage, including reversals of sex-specific effects during ontogeny. In particular, sexual dimorphism was reversed for body size (length of tibia plus patella of the first pair of legs) and body mass, with males being larger but females heavier. We conclude that while females are selected to increase mass and hence fecundity, sexual selection apparently favours larger males (Schaefer and Uhl, 2003) but at the same time earlier maturing males (i.e. protandry; see Schäfer and Uhl, 2002). Achieving both seems only possible when food is plentiful and is facilitated by a low genetic correlation between development time and body size. However, the relative strengths of these various selective pressures, presumably explaining the current sexual size dimorphism in this species, remains to be compared directly. Finally, our results also demonstrated genetic variation, as well as genotype–environment interactions, for all life-history traits examined. Genetic variation was not greater under food stress conditions but genetic covariation was, so this issue remains unresolved (cf. Hoffmann and Merilä, 1999). It would be interesting to generate comparable data for other spider species with female-biased size dimorphism (Elgar, 1992, 1998; Vollrath and Parker, 1992).

ACKNOWLEDGEMENTS

We thank Jutta Schneider and Michael Schmitt for helpful discussions. Bärbel Bauch and Ute Grundtner kindly helped with rearing masses of spiderlings. This study was supported by the Deutsche Forschungsgemeinschaft Uh87/2-2, which we gratefully acknowledge.

REFERENCES

Abrams, P.A., Leimar, O., Nylin, S. and Wiklund, C. 1996. The effect of flexible growth rates on optimal sizes and development times in a seasonal environment. *Am. Nat.*, **147**: 381–395.

Andersson, M. 1994. *Sexual Selection*. Princeton, NJ: Princeton University Press.

Badyaev, A.V., Whittingham, L.A. and Hill, G.E. 2001. The evolution of sexual dimorphism in the house finch. III. Developmental basis. *Evolution*, **55**: 176–189.

Becker, W.A. 1992. *Manual of Quantitative Genetics*, 5th edn. Pullman, WA: Academic Enterprises.

Blanckenhorn, W.U. 1998. Adaptive phenotypic plasticity in growth rate and diapause in the yellow dung fly. *Evolution*, **52**: 1394–1407.

Blanckenhorn, W.U. 2000. The evolution of body size: what keeps organisms small? *Quart. Rev. Biol.*, **75**: 385–407.

Blanckenhorn, W.U. 2002. The consistency of heritability estimates in field and laboratory in the yellow dung fly. *Genetica*, **114**: 171–182.

Blanckenhorn, W.U. and Hosken, D.J. 2003. The heritability of three condition measures in the yellow dung fly. *Behav. Ecol.*, **14**: 612–618.

Clutton-Brock, T.H., Albon, S.D. and Guinness, F.E. 1985. Parental investment and sex differences in juvenile mortality in birds and mammals. *Nature*, **313**: 131–133.

Coddington, J.A., Hormiga, G. and Scharff, N. 1997. Giant female or dwarf male spiders? *Nature*, **385**: 687–688.

David, P., Bjorksten, T., Fowler, K. and Pomiankowski, A. 2000. Condition-dependent signalling of genetic variation in stalk-eyed flies. *Nature*, **406**: 186–188.

Elgar, M.A. 1992. Sexual cannibalism, size dimorphism and courtship behaviour in orb-weaving spiders and other invertebrates. In *Cannibalism: Ecology and Evolution Among Diverse Taxa* (M.A. Elgar and B.J. Crespi, eds.), pp. 128–155. Oxford: Oxford University Press.

Elgar, M.A. 1998. Sperm competition and sexual selection in spiders and other arachnids. In *Sperm Competition and Sexual Selection* (T.R. Birkhead and A.P. Moller, eds.), pp. 307–339. San Diego, CA: Academic Press.

Fairbairn, D.J. 1997. Allometry for sexual size dimorphism: pattern and process in the coevolution of body size in males and females. *Annu. Rev. Ecol. Syst.*, **28**: 659–687.

Falconer, D.S. 1989. *Introduction to Quantitative Genetics*. Harlow, UK: Longman Scientific & Technical.

Fischer, K. and Fiedler, K. 2000. Sex related differences in reaction norms in the butterfly *Tityrus*. *Oikos*, **90**: 372–380.

Fischer, K. and Fiedler, K. 2001. Dimorphic growth patterns and sex-specific reaction norms in the butterfly *Lycaena hippothoe sumadiensis*. *J. Evol. Biol.*, **14**: 210–218.

Fraser, D.F. and Gilliam, J.F. 1992. Nonlethal impacts of predator invasion: facultative suppression of growth and reproduction. *Ecology*, **73**: 959–970.

Fritz, R.S. and Morris, D.H. 1985. Reproductive success and foraging of the crab spider *Misumena vatia*. *Oecologia*, **12**: 194–200.

Fry, J.D. 1992. The mixed-model analysis of variance applied to quantitative genetics: biological meaning of the parameters. *Evolution*, **46**: 540–550.

Gotthard, K., Nylin, S. and Wiklund, C. 1994. Adaptive variation in growth rate: life history costs and consequences in the speckled butterfly. *Oecologia*, **99**: 281–289.

Grafen, A. 1990. Biological signals as handicaps. *J. Theor. Biol.*, **144**: 517–546.

Higgins, L.E. and Rankin, M.A. 1996. Different pathways in arthropod postembryonic development. *Evolution*, **50**: 573–582.

Hoffmann, A.A. and Merilä, J. 1999. Heritable variation and evolution under favourable and unfavourable conditions. *Trends Ecol. Evol.*, **14**: 96–101.

Hoffmann, A.A. and Parsons, P.A. 1991. *Evolutionary Genetics and Environmental Stress*. Oxford: Oxford University Press.

Homann, H. 1949. Über das Wachstum und die mechanischen Vorgänge bei der Häutung von *Tegenaria agrestis* (Araneae). *Zeitsch. vergl. Physiol.*, **31**: 413–440.

Honêk, A. 1993. Intraspecific variation in body size and fecundity in insects: a general relationship. *Oikos*, **66**: 483–492.

Houle, D. 1992. Comparing evolvability and variability of quantitative traits. *Genetics*, **130**: 195–204.

Hutchinson, J.M., McNamara, J.M., Houston, A.I. and Vollrath, F. 1997. Dyar's rule and the investment principle: optimal moulting strategies if feeding rate is size-dependent and growth is discontinuous. *Phil. Trans. R. Soc. Lond. B*, **352**: 113–138.

Jakob, E.M. and Dingle, H. 1990. Food level and life history characteristics in a pholcid spider (*Holocnemus pluchei*). *Psyche*, **97**: 95–110.

Jakob, E.M., Marshall, S.D. and Uetz, G.W. 1996. Estimating fitness: a comparison of condition indices. *Oikos*, **77**: 61–67.

Kessler, A. 1971. Relation between egg production and food consumption in species of the genus *Pardosa* (Lycosidae, Araneae) under experimental conditions of food abundance and food shortage. *Oecologia*, **8**: 93–109.

Klingenberg, C.P. 1998. Individual variation of ontogenies: a longitudinal study of growth and timing. *Evolution*, **50**: 2412–2428.

Kozlowski, J. 1992. Optimal allocation of resources to growth and reproduction: implications for age and size at maturity. *Trends Ecol. Evol.*, **7**: 15–19.

Mayntz, D. and Toft, S. 2001. Nutrient composition of the prey's diet affects growth and survivorship of a generalist predator. *Oecologia*, **127**: 207–213.

Miyashita, K. 1988. Egg production in *Pholcus phalangioides* (Fuesslin) (Araneae, Pholcidae) under a constant temperature and photoperiod. *J. Arachnol.*, **16**: 129–131.

Mousseau, T.A. and Roff, D.A. 1987. Natural selection and the heritability of fitness components. *Heredity*, **59**: 181–197.

Nylin, S. and Gotthard, K. 1998. Plasticity in life history traits. *Annu. Rev. Entomol.*, **43**: 63–83.

Peterson, B. 1950. The relation between size of mother and number of eggs and young in some species and its significance for the evolution of size. *Experientia*, **6**: 96–98.

Pomiankowski, A. and Møller, A.P. 1995. A resolution to the lek paradox. *Proc. R. Soc. Lond. B*, **260**: 21–29.

Prenter, J., Elwood, R.W. and Montgomery, W.I. 1998. No association between sexual size dimorphism and life histories in spiders. *Proc. R. Soc. Lond. B*, **265**: 57–62.

Preziosi, R. and Roff, D.A. 1998. Evidence of genetic isolation between monomorphic and sexually dimorphic traits in the water strider *Aquarius remigis*. *Heredity*, **81**: 92–99.

Roff, D.A. 1980. Optimizing development time in a seasonal environment: the 'ups and downs' of clinal variation. *Oecologia*, **45**: 202–208.

Roff, D.A. 1992. *The Evolution of Life Histories*. New York: Chapman & Hall.

Roff, D.A. 1995. The estimation of genetic correlations from phenotypic correlations: a test of Cheverud's conjecture. *Heredity*, **74**: 481–490.

Roff, D.A. 1996. The evolution of genetic correlations: an analysis of patterns. *Evolution*, **50**: 1392–1403.

Roff, D.A. 1997. *Evolutionary Quantitative Genetics*. New York: Chapman & Hall.

Roff, D.A. 2000. Trade-offs between growth and reproduction: an analysis of the quantitative genetic evidence. *J. Evol. Biol.*, **13**: 434–445.

Roff, D.A. and Preziosi, R.F. 1994. The estimation of the genetic correlation: the use of the jackknife. *Heredity*, **73**: 544–548.

Rowe, L. and Houle, D. 1996. The lek paradox and the capture of genetic variance by condition dependent traits. *Proc. R. Soc. Lond. B*, **263**: 1415–1421.

Rowe, L. and Ludwig, D. 1991. Size and timing of metamorphosis in complex life cycles: time constraints and variation. *Ecology*, **72**: 413–427.

Schaefer, D. and Uhl, G. 2003. Male competition over access to females in a spider with last-male sperm precedence. *Ethology*, **109**: 385–400.

Schaefer, M. 1976. Experimentelle Untersuchungen zum Jahreszyklus und zur Überwinterung von Spinnen (Araneida). *Zool. Jb. Syst.*, **103**: 127–289.

Schäfer, M.A. and Uhl, G. 2002. Determinants of paternity success in the spider *Pholcus phalangioides* (Pholicidae: Araneae): the role of male and female mating behaviour. *Behav. Ecol. Sociobiol.*, **51**: 368–377.

Shine, R. 1988. The evolution of large body size in females: a critique of Darwin's fecundity advantage model. *Am. Nat.*, **131**: 124–131.

Signorelli, M. 2002. Does sexual size dimorphism imply a corresponding development time difference? Diploma thesis, University of Zürich.

Simpson, M.R. 1993. Reproduction in two species of arctic arachnids, *Pardosa glacialis* and *Alopecosa hirtipes*. *Can. J. Zool.*, **71**: 451–457.

Simpson, M.R. 1995. Covariation of spider egg and clutch size: the influence of foraging and parental care. *Ecology*, **76**: 795–800.

Skow, C.D. and Jakob, E.M. 2003. Effect of maternal body size on clutch size and egg weight in a pholcid spider (*Holocnemus pluchei*). *J. Arachnol.*, **31**: 305–308.

Sokal, R.R. and Rohlf, F.J. 1995. *Biometry: The Principles and Practice of Statistics in Biological Research*. New York: Freeman.

Stearns, S.C. 1992. *The Evolution of Life Histories*. Oxford: Oxford University Press.

Stearns, S.C. and Koella, J. 1986. The evolution of phenotypic plasticity in life history traits: predictions of reaction norms for age and size at maturity. *Evolution*, **40**: 893–914.

Uetz, G.W., Bischoff, J. and Raver, J. 1992. Survivorship of wolf spiders (Lycosidae) reared on different diets. *J. Arachnol.*, **20**: 207–211.

Uhl, G. 1994a. Reproduktionsbiologie von Zitterspinnen (*Pholcus phalangioides*; Pholcidae; Araneae). PhD thesis, University of Freiburg, Germany.

Uhl, G. 1994b. Internal courtship in cellar spiders (*Pholcus phalangiodes*). *Verhandl. Deutsch. Zool. Ges.*, **87.1**: 64.

Uhl, G. 1998. Mating behaviour in the cellar spider, *Pholcus phalangioides*, indicates sperm mixing. *Anim. Behav.*, **56**: 1155–1159.

Vertainen, L., Alatalo, R.V., Mappes, J. and Parri, S. 2000. Sexual differences in growth strategies of the wolf spider *Hygrolycosa rubrofasciata. Evol. Ecol.*, **14**: 595–610.

Via, S. 1984. The quantitative genetics of polyphagy in an insect herbivore. II. Genetic correlations in larval performance within and among host plants. *Evolution*, **38**: 896–905.

Vollrath, F. 1987. Growth, foraging and reproductive success. In *Ecophysiology of Spiders* (W. Nentwig, ed.), pp. 357–370. Berlin: Springer.

Vollrath, F. and Parker, G.A. 1992. Sexual dimorphism and distorted sex ratios in spiders. *Nature*, **360**: 156–159.

Werner, E.E. and Anholt, B.R. 1993. Ecological consequences of the trade-off between growth and mortality rates mediated by foraging activity. *Am. Nat.*, **142**: 242–272.

Wiklund, C. and Fagerström, T. 1977. Why do males emerge before females? A hypothesis to explain the incidence of protandry in butterflies. *Oecologia*, **31**: 153–158.

Zonneveld, C. 1996. Being big or emerging early? Polyandry and the trade-off between size and emergence in male butterflies. *Am. Nat.*, **147**: 946–965.